# Current Status of Intraaortic Balloon Pump and Initial Clinical Experience With Aortic Patch Mechanical Auxiliary Ventricle

By Adrian Kantrowitz, Joseph S. Krakauer, George Zorzi, Melvyn Rubenfire, Paul S. Freed, Steven Phillips, Marc Lipsius, Claudio Titone, Philip Cascade, and Dov Jaron

The purpose of this report is to summarize our laboratory's experience with two promising modalities of in-series circulatory assistance: the intraaortic phase-shift balloon pump and the dynamic aortic patch, a permanently implanted mechanical auxiliary ventricle. Our clinical experience with 40 patients in medically refractory cardiogenic shock who received circulatory assistance by balloon pumping is summarized, and preliminary observations on the initial clinical experience with the permanent assist device is presented.

## Temporary In-Series Ventricular Support: Phase-Shift Balloon Pumping

Phase-shift balloon pumping is a type of in-series circulatory assistance in which a flexible pumping chamber situated within the thoracic aorta is expanded and deflated synchronously and out of phase with the heart. This type of assisted circulation thus introduces periodic pressure perturbations into the central aorta at the same frequency as the heart rate. Its action may be represented as change in the afterload on the left ventricle[14] is illustrated in Fig. 1 showing a recording of hemodynamic parameters from a canine experiment. The variables include aortic root pressure, its fundamental component (obtained by low

pass filtering), aortic root flow, and its fundamental component. When the balloon is inactive (left portion of tracing), the fundamental components of pressure and flow waveforms are approximately in phase (the maxima and minima of the waves occur approximately at the same time). When the balloon is activated, the fundamental components of pressure and flow are approximately 180° apart (i.e., the maximum of one wave occurs at a time when the other wave is at a minimum). Consequently, the fundamental component of the afterload impedance (pressure/flow) has a phase angle of approximately 180°. Thus, the effect of pumping is to shift the phase of the aortic root impedance. The relationship between phase angle during pumping and hemodynamic benefits of assistance will be discussed below.

### Apparatus and Method

The principle of balloon pumping was first reported by Moulopoulos et al.[27] and by Clauss et al.[5] in 1962. At that time a practicable system was not developed. In 1967, our laboratory presented experimental results obtained with a different configuration of the apparatus.[34] This system[21,22,34] utilized a nonocclusive flexible polyurethane balloon mounted on a polyurethane catheter (Fig. 2). The volume of the inflated balloon is 33 cc, approximately equal to the stroke volume of the failing left ventricle. The balloon is threaded through a sleeve of Dacron arterial graft into a femoral arteriotomy to the thoracic aorta until its tip is approximately at the

AORTIC ROOT PRESSURE

FUNDAMENTAL COMPONENT OF
AORTIC ROOT PRESSURE

AORTIC ROOT FLOW

FUNDAMENTAL COMPONENT OF
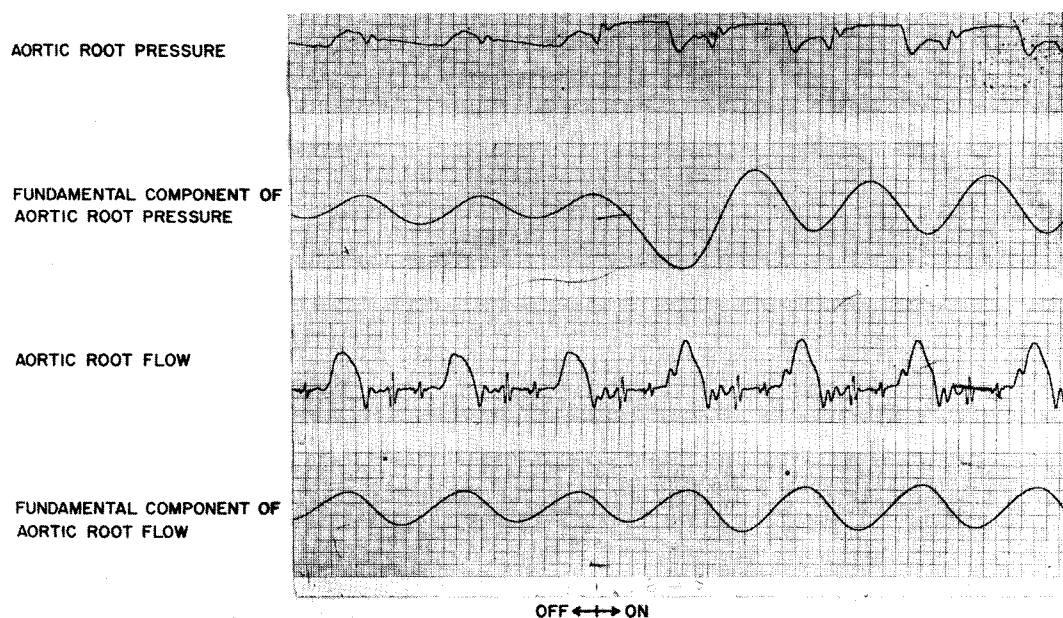AORTIC ROOT FLOW

OFF ←—+—→ ON

Fig. 1.—Recording of aortic root pressure and flow and their fundamental components before (left portion of tracing) and during intraaortic balloon pumping in dog. Flow signal is transmitted virtually instantaneously; pressure signal is delayed approximately 30–40 msec because of mechanical inertia of fluid in pressure catheter, catheter length and elasticity, and transducer. Fundamental components of pressure and flow appear to be not precisely in phase when balloon is inactive.

level of the left subclavian artery. The sleeve is then sutured to the femoral artery incision. This permits release of the occlusive snares around the vessel and restores circulation to the extremity.[19]

The distal end of the catheter is attached to the pneumatic port of an electronic control unit (Fig. 3) also developed in our laboratory.* The control unit is triggered by signals derived from the R-wave of the electrocardiogram to operate a fast-acting, three-way solenoid valve that admits helium at a pressure of 120–150 mm Hg to the catheter and exhausts it to atmosphere. The action of the solenoid valve is controlled by two adjustable time delays so as to inflate the balloon at the beginning of ventricular diastole and to deflate it at the beginning of systole. The control unit de-

flates the balloon automatically when a premature beat is sensed.

### Experimental Observations

Beneficial hemodynamic effects of diastolic augmentation as a result of elevation of the diastolic pressure in the aorta were reported in 1953 by Kantrowitz and Kantrowitz.[17] Following this demonstration, a number of techniques based on this principle have been studied.[4,11,16,20,31,37]

In animal experiments performed in our laboratory,[14,15,34,42,45] a number of facets of hemodynamic responses to phase-shift pumping have been defined (Fig. 4). In animals with induced heart failure by serial coronary artery ligation, the following average changes due to circulatory assistance by means of balloon pumping have been demonstrated: Left ventricular end-diastolic pressure was reduced by 40% and tension-time index by 20%. Cardiac

*These balloon pumps and control units are distributed by the Milton Roy Co., St. Petersburg, Fla.

Fig. 3.—Control unit for phase-shift pumping. In addition to operation in automatic mode, inflation and deflation can be manually controlled as adjunct to resuscitation after cardiac arrest.

output, coronary blood flow, and left ventricular dp/dt were increased by 50%, 100%, and 25%, respectively. Reduction in left ventricular peak, and end-diastolic pressures, and tension-time index reflect a decrease in myocardial wall tension and suggest a reduction in myocardial oxygen demand. The reduced end-diastolic pressure suggests diminished left ventricular volume and thus reversal of compensatory cardiac dilatation secondary to failure. The increase in central aortic diastolic pressure enhances coronary artery perfusion pressure. These effects, in conjunction with decreased systolic ejection time and diminished myocardial wall tension, produce increased coronary flow and myocardial oxygenation.

These observations have been corroborated and extended in a number of other investigations. In an early study, Brown et al.[2] found that balloon pumping caused favorable changes in hemodynamic parameters of dogs in experimental left ventricular failure and extended their period of survival. Powell et al.[32] found that in hypotensive canine right-heart bypass preparations, the results of balloon pumping included an increase of coronary blood
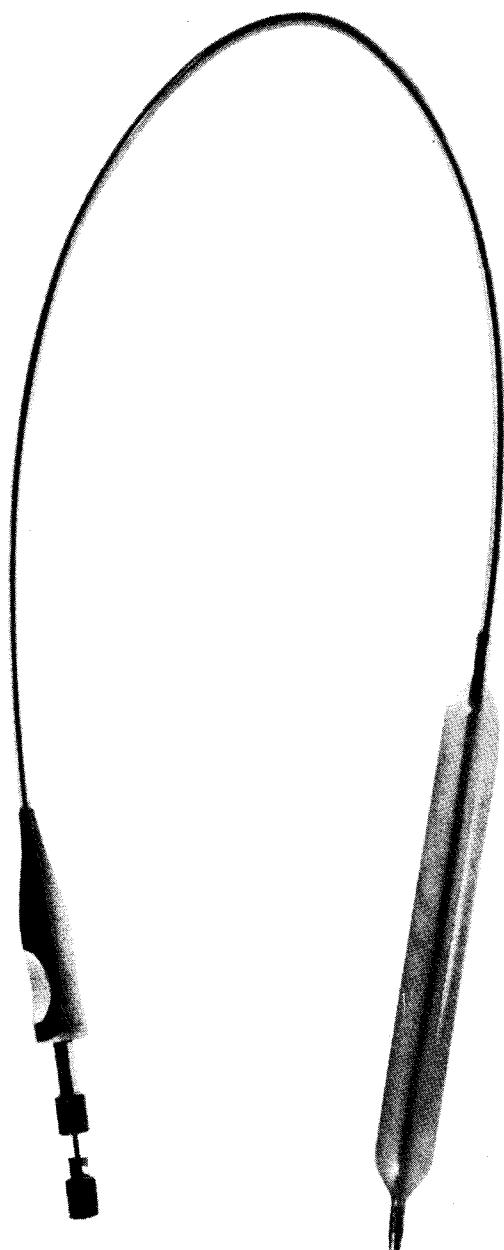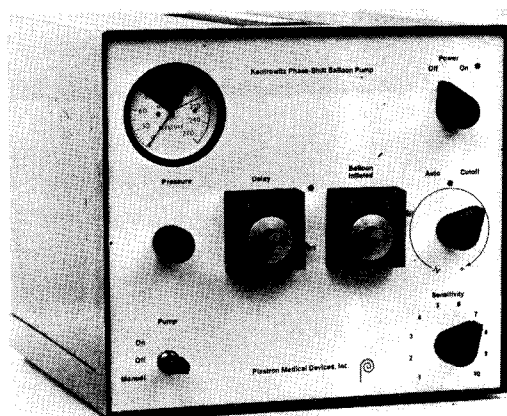
Fig. 2.—Phase-shift balloon pump. Polyurethane of which balloon is fabricated is exposed to transmembrane pressures of 50 mm Hg during operation but can withstand 300 mm Hg without undergoing elastic deformation. Markedly higher pressures are required to burst balloon. In accelerated life tests, balloons have been operated for 15,000,000 cycles at 200 mm Hg pressure without failure.
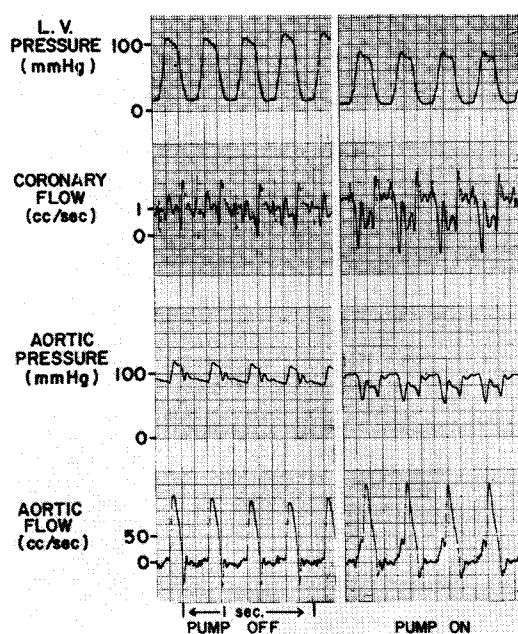
Fig. 4.—Recording of hemodynamic measurements in dog with experimental heart failure before and during balloon pumping. Phasing of pump and cardiac cycles is one of critical determinants of effectiveness of pumping.

flow of 40.9 ± 8.6 ml/min associated with an increment of $MV_{O2}$ of 1.2 ± .03 ml/min/100 g LV, a decrease of left ventricular peak systolic pressure of −5±1 mm Hg, and reduction toward normal (−9.9 ± 1.6 cm $H_2O$) of elevated left ventricular end-diastolic pressure. They concluded that balloon pumping can decrease left ventricular peak systolic pressure and end-diastolic pressure independently of changes in coronary blood flow and that a major effect of mechanical assistance in the hypotensive, coronary flow-limited preparation is improvement in cardiac efficiency.

Talpins et al.[40] compared balloon pumping and counterpulsation in dogs with cardiogenic shock induced by serial coronary artery ligation. Hemodynamic effects of balloon pumping included decrease in the left ventricular mean systolic pressure (26.6%), left ventricular stroke work

(11.5%), maximal dp/dt (46.4%), ventricular work/min (28.8%), and tension-time index (35.4%), and increase in coronary blood flow (20%). Mean decrease in cardiac output of 8.2% was not considered significant. They concluded that balloon pumping reduced left ventricular work to the same extent as counterpulsation and was preferable for clinical use because of the simplicity of its application.

Corday et al.[6] reported favorable effects of balloon pumping in six dogs with hypotension induced by serial ligation of branches of the left circumflex coronary artery. Their results showed that flow in the left anterior descending coronary artery increased 12.8%, left ventricular peak systolic pressure decreased 8.6%, and ventricular end-diastolic pressure was reduced 25.4% from controls. They also reported increased renal resistance and reduced renal blood flow.

Tyberg et al.[43] used a canine right heart bypass preparation to produce low levels of cardiac output. To minimize bubble blowing, a three-segment balloon was used. They concluded that although their experimental model did not duplicate the elevated left ventricular end-diastolic pressure and cardiac dilatation of cardiogenic shock, balloon pumping improved the performance of hearts that were underperfused and probably locally ischemic. Results of another investigation reported by the same group[44] indicated that prior to coronary artery ligation, balloon pumping decreased left ventricular systolic peak pressure and increased aortic diastolic pressure. Peak aortic flow, stroke volume, and cardiac output were all augmented. Smaller changes due to cardiac assistance were observed after coronary artery ligation. The investigators concluded that adequate systolic ejection is essential for effective cardiac assistance.

A number of studies have failed to disclose beneficial effects of intraaortic balloon pumping.[8,10,24,41] A recent series of

experiments in our laboratory by Jaron et al. may help to account for such findings.[14] The purpose of this investigation was to examine the relationship between the hemodynamic effectiveness of assistance and the adjustment of the times of balloon inflation and deflation. This adjustment is subjective and difficult to reproduce when based on visual inspection of the central aortic pressure waveform. Balloon pumping was performed at various settings of inflation and deflation before and after induction of experimental cardiogenic shock. The phase angle of the afterload was monitored. The results indicated that most hemodynamic benefits of assistance were strongly dependent on the left ventricular afterload phase angle, which varies according to the adjustment of balloon inflation and deflation relative to the cardiac cycle (Fig. 1). Improvement in coronary flow, external left ventricular work, work in the aorta, and tension time index was maximal when the afterload phase angle was approximately 180°. Small deviations from 180° were associated with large reductions in hemodynamic benefits. Large deviations from 180° caused changes in some hemodynamic parameters opposite to those desired. Although data for cardiac output followed a parallel trend, the relationship to afterload phase angle was not as clear-cut as for the other variables.

An additional observation in this study was that an afterload phase angle of 180° could not be achieved when a balloon with a maximal diameter larger than that of the aorta was used. Hemodynamic effects under this condition were significantly reduced or even lost.

The results indicated that a left ventricular afterload phase angle of approximately 180° is a necessary condition for maximal effectiveness of balloon pumping and other modalities of in-series circulatory assistance. Thus, the conclusions of investigations in which negligible hemodynamic benefits of balloon pumping were reported may require reassessment in the light of the above findings, as well as other factors enumerated in the report by Jaron et al.[14]

## Clinical Observations

Clinical application has confirmed some of the experimental observations on phase-shift pumping and has defined other physiologic responses to the procedure. Our initial work with the method was restricted to patients in medically refractory cardiogenic shock secondary to acute myocardial infarction. Since methods for control of arrhythmias and congestive failure have improved in recent years, the importance of shock as a cause of death in acute myocardial infarction has increased. Mortality in cardiogenic shock has not been determined in a large, prospectively studied population, although the MIRU (Myocardial Infarction Research Units, National Heart and Lung Institute, USPHS) investigations now being conducted may be expected eventually to provide this information. In smaller individual series of cases, the incidence of fatal outcome has varied from 85 to 100%.[7,9,33]

The pathophysiologic abnormalities in intractable cardiogenic shock have not been completely defined and quantitated. Therefore, in selecting candidates for assisted circulation, we have had to utilize the generally accepted clinical manifestations as a basis for the diagnosis. At transfer to the cardiac assistance group, candidates for balloon pumping had classic symptoms and signs of intractable cardiogenic shock: systolic blood pressure of less than 80 mm Hg (during interruption of vasopressor therapy), cold, clammy skin reflecting generalized sympathetic hyperactivity, and urine flow of 10 ml/hr or less, with or without mental obtundation.

Balloon pumping, shown to be effective in initial animal experiments, was attractive for the treatment of cardiogenic shock because of its ability to augment coronary flow, and to decrease of left ventricular

external work, thereby reducing myocardial oxygen requirements while maintaining adequate systemic perfusion. These effects suggested that the technique might serve as a means of interrupting the progressive cardiovascular deterioration and loss of homeostatic control mechanisms in shock. At the same time, it appeared to us that patients in shock unresponsive to vigorous medical management offered an appropriate population in which to test the clinical efficacy of balloon pumping and to identify the possible complications of the method.

Our experience in treating 40 patients in intractable cardiogenic shock has been presented in part in several previous reports.[18,21-23] Patients were referred for pumping when the probability of reversal of the shock state appeared negligible despite maximal application of pharmacologic therapy.

Definitive clinical management during balloon pumping has not been established, but the effectiveness of certain tentative guidelines is suggested by our observations in these cases.[23] On initiation of pumping, heparin is administered and repeated at regular intervals. Efforts are directed toward improving cardiac function and correction of systemic abnormalities due to the shock state and its prior treatment: volume abnormalities, hyponatremia, hemodilution, hypoxemia, and acid-base aberrations that often are masked by defective myocardial contractility.

Vasopressor treatment is withdrawn, if possible, when the assist device has been placed in the vascular system and optimally synchronized with the cardiac cycle[14] (Fig. 4). In instances when mean central aortic pressure is below 50 mm Hg and evidence of occlusion of the aorta by the inflated balloon is present, administration of pressor amines is continued for a brief period. Pressor support is stopped when cardiac assistance alone is capable of producing central aortic pressures sufficient to main-

tain an aortic diameter larger than that of the inflated balloon.

In cases where satisfactory central aortic pressures were obtained but diaphoresis and oliguria tended to persist and capillary filling remained poor, a vasodilating agent, chlorpromazine, was given in a dosage of 0.1–0.2 mg/kg except to patients thought to be in right heart failure. Digitalis was given to all patients who demonstrated evidence of congestive heart failure.

When adequate perfusion had been achieved (as evidenced by restoration of urine flow, warming of the extremities, and improved sensorium), abnormalities of effective plasma volume, pH, serum osmolarity, and red cell mass were corrected.[23] Functional hypovolemia, hyponatremia, and metabolic acidosis were frequently seen in these patients, but occult right ventricular failure also occurred. In such cases phlebotomy was performed, despite normal central venous pressure and hypotension. Hemodilution due to phlebotomy was corrected by infusion of packed red cells.

Severe hypoxemia was observed in all patients and in most resisted conventional treatment. When necessary, patients were intubated and ventilation was assisted or controlled with a volume cycled respirator. Adequate management of this problem has not been evolved yet, and further study is needed.

Pumping was sustained until shock was clinically reversed and all metabolic aberrations had been corrected. If several hours' additional pumping produced no change in the patient's condition, mechanical assistance was interrupted, and if no immediate deterioration was noted, the procedure was discontinued for 30 min. Pumping was then resumed for 30 min. If the patient's condition improved during this period, pumping was continued for several hours more before trial discontinuation was again attempted. If the 30-min trial pumping caused no change in the

patient's state, pumping was terminated. The balloon was left in situ for 24 hr, and if the patient remained stable, it was then removed.

Just as positive criteria for discontinuing assistance have not been identified, no definite limitations to its duration have been established. Whereas only a few hours were required for restoration of circulatory stabilization in some cases, in others 2–3 days and more were needed. In the longest duration of pumping recorded to date by Wolff et al. at the Barnes Hospital in St. Louis, a patient was supported, with only brief interruptions, by balloon pumping over a period of 20 days (G. Wolff, personal communication). The patient did not survive.

In our series of cases treated at Maimonides Hospital, Brooklyn, N.Y., and Sinai Hospital of Detroit, the results which have been described in part elsewhere,[3,18,21,22,23] were as follows. Twenty-five patients had onset of cardiogenic shock soon after myocardial infarction, in most cases, within 12 hr of the acute infarct, and in all, within 30 hr. During pumping, symptoms of shock were reversed in 23 or 90%. Nineteen patients, or 75%, regained hemodynamic stabilization, enabling termination of mechanical assistance. The remaining six patients died during pumping.

Of the 19 patients who recovered from cardiogenic shock, 11 were discharged from the hospital on convalescing from their infarcts. Three of these patients died, 19, 24, and 7 mo, respectively, following mechanical assistance. The remainder were well 5–47 mo after treatment. The remaining nine patients died 8 hr–7 days after pumping.

In 15 patients in this series, the onset of shock was delayed 30 hr or longer after acute myocardial infarction. Shock was clinically reversed during pumping in 12 patients. Six died during pumping, and thus only nine patients regained hemodynamic stabilization that allowed termi-

nation of mechanical assistance. Death in these cases occurred 12 hrs–6 wk later.

Myocardial rupture was seen in one patient with early shock, and in seven of those with delayed circulatory collapse.

Complications due to balloon pumping have been relatively minor: two instances of infection at the site of the femoral arteriotomy,[18] and, in the first patient, in whom the Dacron side-arm graft was not used, circulatory insufficiency below the arterial incision.[19] Temporary neuropathic complications with flaccid paralysis and absent deep tendon reflexes at the knee and ankle occurred in a recent long-term survivor; 3 mo after the procedure the patient has completely recovered. Utilizing studies of red cell morphology, serum haptoglobin reticulocyte count, plasma hemoglobin, serum bilirubin, and platelet counts, no significant hemolysis or other damage to formed elements of the blood has been detected in any patient in this series.

Although the frequency of myocardial rupture has been higher than that usually expected in patients with acute myocardial infarction, we do not believe that it constitutes a complication of mechanical assistance. Since the aortic valve is closed during inflation of the balloon, and since the action of the device is to reduce left ventricular peak systolic pressure and thereby myocardial wall tension, it seems unlikely that mechanical assistance causes myocardial rupture. A more reasonable explanation may be that pumping prolongs survival of patients with massive myocardial insults long enough to allow the complete pathophysiologic sequence of events to unfold. This matter and the apparent difference in prognosis according to the interval from infarct to onset of shock need to be investigated further.

Other groups have also reported clinical studies of balloon pumping in patients with cardiogenic shock after acute myocardial infarction.[26,28-30,39] Participants in the Cooperative Study: Phase-shift Bal-

loon Pumping in Cardiogenic Shock, using the equipment developed by our group, are also accumulating clinical data.[28,29,39]

Leinbach et al.[26,30] treated 10 patients in severe cardiogenic shock after myocardial infarction. They found that assisted circulation resulted in striking clinical and hemodynamic improvement. Bregman et al.[1] treated four patients in cardiogenic shock with a dual-chambered balloon. One survived. An additional patient in intractable left ventricular failure was assisted effectively in preparation for cardiac surgery.

PERMANENT IN-SERIES VENTRICULAR SUP-
PORT: MECHANICAL AUXILIARY VENTRICLE

### Experimental Background

Extensive experimental work on various configurations of a permanently implantable auxiliary ventricle has been carried out in our laboratory. In 1966, a U-shaped mechanical auxiliary ventricle was implanted in two patients. This experience demonstrated the clinical feasibility and the hemodynamic effectiveness of this form of mechanical assistance for patients in chronic left ventricular failure. The demise of the second patient due to a thromboembolism 12 days postoperatively impelled us to reconsider the geometry of the prosthesis and to search for an improved material for the blood interface.[16,20]

The new configuration, the dynamic aortic patch, is based on the same principle as the balloon pump. Since this prosthesis was to be implanted into the wall of the descending aorta, it would interfere minimally with the hydrodynamics of blood flow. Only one artificial material had to be used for the intravascular interface. We explored the possibility of using a material that would favor tissue ingrowth, leading to a blood interface that would eventually be covered by a pseudointima. This would be expected to provide a biological interface having a low potential for thromboembolism.
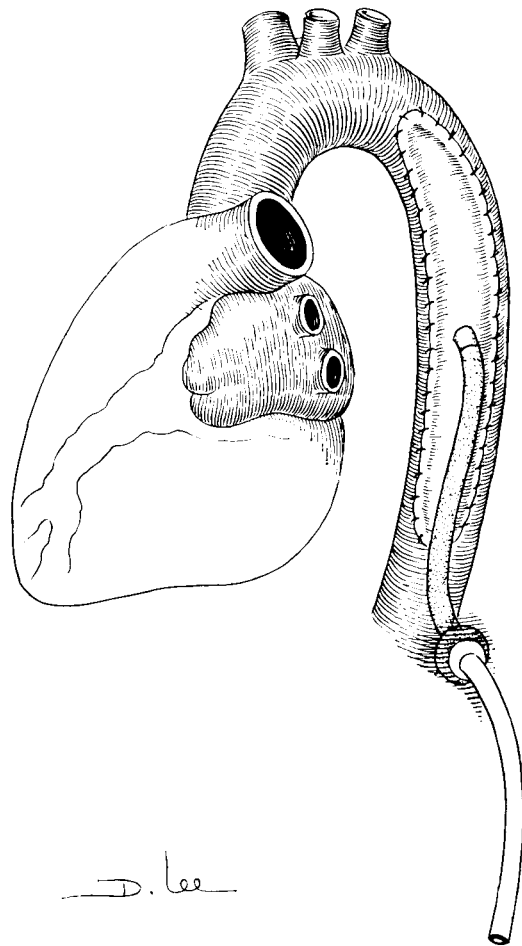


Fig. 5.—Dynamic aortic patch in situ.

In its present configuration the dynamic aortic patch (Fig. 5) consists of an elliptical silicone rubber pumping chamber, covering materials, and a gas conduit. The covering material used for the intravascular surface consists of Dacron velour backed with a conductive polyurethane.[35,36] Plain Dacron cloth is used for the outer surface. The prosthesis is implanted on the lateral surface of the descending thoracic aorta between the origin of the left subclavian artery and the diaphragm. Double rows of continuous everting sutures reinforced with Dacron felt are used to suture the dynamic aortic patch into the vessel wall. The gas

conduit is led to a transcutaneous connector implanted in the left hypogastric area. The transcutaneous connector was built according to the findings reported by Lee et al.[25] ECG electrodes implanted in the left ventricular myocardium are also brought to the transcutaneous connector. An extracorporeal driving system is attached to the prosthesis through the connector. The driving system consists either of the electropneumatic driver used for balloon pumping or a portable, battery-driven compressor built into a vest. Both systems utilize the R wave of the electrocardiogram to trigger the pump, as for balloon pumping.

A report of preliminary studies of this auxiliary ventricle was presented by Sujansky et al.,[38] and detailed accounts of subsequent studies using different materials for the prosthesis are in preparation. Results of experiments utilizing Dacron velour backed with conductive polyurethane for the blood interface showed that in 84 dogs in which inactive prostheses had been implanted, an organized layer of pseudointima covered most or all of the artificial material. Four to six months were required for complete coverage, but a stable layer of fibrin was deposited within hours after the surgical procedure. In 26 experiments in which the prosthesis was activated immediately after implantation and intermittently thereafter, data were obtained suggesting that pumping delayed but did not prevent formation of the pseudointimal layer. Hematologic studies disclosed no evidence of hemolysis or gross damage to formed elements of the blood. A wedge-shaped, 0.08 × 0.05 × 0.05-cm, yellow-white infarct was found in the cortex of one kidney in one animal.

The hemodynamic effects of the dynamic aortic patch were evaluated in 19 studies in ten dogs before and during periods of pumping.[13] In these studies, each animal served as its own control. The results (Table 1) indicated that left ventricular peak pressure, tension-time index, stroke

Table 1.—Hemodynamic Effects of Dynamic Aortic Patch in Dogs in Experimental Left Ventricular Failure

| Parameter | Mean Per Cent Change |
|---|---|
| Left ventricular peak pressure | −23 |
| Tension-time index | −23 |
| Left ventricular stroke work | −18 |
| Systemic vascular resistance | −32 |
| Cardiac output | +16 |
| Left circumflex coronary artery blood flow | +41 |
| Peak diastolic pressure at aortic root | +19 |
| Mean systolic ejection rate | +36 |

Each animal served as its own control in these experiments. Mean per cent change: change from unassisted to assisted condition. All changes from control values were significant at 0.01 level (Student's t test).

work, and systemic vascular resistance, all decreased significantly when the dynamic aortic patch was activated. Concurrently, there were substantial improvements in cardiac output, left circumflex coronary blood flow, diastolic blood pressure, and mean systolic ejection rate. These effects were comparable to those produced by balloon pumping and previous configurations of the mechanical auxiliary ventricle.

Clinical Experience

In view of this evidence of the hemodynamic effectiveness and freedom from thromboembolic potential of the dynamic aortic patch, we decided that a limited clinical trial of the system was appropriate in patients with severe, intractable heart failure unresponsive to conventional therapy. A patient meeting these criteria was recently referred to us.

He was a 63-yr-old man with chronic congestive heart failure unresponsive to medical treatment. He was bedridden because of weakness, severe dyspnea, and recurrent leg edema. Symptoms had first been noted in early 1967 when the patient developed shortness of breath, two-pillow orthopnea, and leg edema. A conservative medical regimen consisting of digitalis and

Table 2.—Initial Cardiac Catheterization of
Patient (May 11, 1971)

| Pressure (mm Hg) Systolic/Diatolic Mean | | | Oxygen Saturation (%) |
|---|---|---|---|
| PCW | | 29 | |
| LPC | | 27 | |
| MPA | 64/22 | 37 | 58 |
| RV | 64/10 | | |
| RA | | 8 | 62 |
| LV | 90/23° | | 86 |
| AO | 90/55 | 67 | |
| Cardiac output | 4.7 liters/min | | |
| Cardiac index | 2.2 liters/min/sq m | | |
| O₂ consumption | 254 cc/min | | |
| Pulmonary arteriolar resistance index | 327.2 dynes/sec/cm⁻⁵/sq m | | |
| Total pulmonary resistance index | 1344 dynes/sec/cm⁻⁵/sq m | | |
| Total peripheral resistance index | 2432 dynes/sec/cm⁻⁵/sq m | | |
| LV work index | 1.31 kg-m/min/sq m | | |

* Left ventricular end diastolic pressure = 23.

PCW: pulmonary capillary wedge; LPC: left
pulmonary capillary wedge; MPA: main pulmo-
nary artery; RV: right ventricle; RA: right atrium;
LV: left ventricle; AO: aortic pressure.

diuretics provided moderate relief until
1969 when the patient complained of pal-
pitations. At that time, an electrocardio-
gram revealed a complete left bundle
branch block, first-degree atrioventricular
block, and frequent premature ventricular
contractions. Subsequently, he was hospital-
ized five times with episodes of pulmonary
edema and severe peripheral edema. From
1967 to August 1971, progressive enlarge-
ment of the heart (140–190 mm transverse
diameter) as well as persistent Kerley B
lines, pleural effusion, and pulmonary vas-
cular congestion, were noted in chest X
rays. After retirement, his condition stead-
ily deteriorated during 1970 in spite of
large dosages of Lasix, Aldactone, Lanoxin,
and Quinidine. Right and left cardiac
catheterization, coronary arteriography, and
left ventriculography were performed else-
where on May 28, 1971 (Table 2). The
results disclosed severe left and right
ventricular failure, severe pulmonary hyper-
tension, enlarged, dilated, poorly con-
tracting left ventricle with an ejection

fraction estimated at 30%, 1+ mitral
regurgitation and greater than 70% narrow-
ing of the main right, main left, and an-
terior descending coronary arteries. Be-
cause of the overwhelming evidence of
cardiac decompensation and the absence of
an anginal syndrome or clear cut myo-
cardial infarction history, the coronary
artery disease was considered inoperable.
A permanent transvenous pacemaker was
implanted in the right ventricle to control
premature ventricular contractions. At the
time of referral, medications included
Lanoxin, Quinidine, Lasix, Aldactone, and
potassium chloride. Clinical evaluation
revealed the following diagnoses: cardio-
myopathy of unknown etiology, athero-
sclerotic heart disease, N. Y. Heart Associa-
tion classification, IVE, diabetes mellitus,
probable cardiac cirrhosis, and mild organic
brain syndrome.

To determine whether this patient would
be a suitable candidate for implantation of
the permanent dynamic aortic patch, we
documented his response to in-series
cardiac assistance by intraaortic balloon
pumping. Table 3 documents the hemo-
dynamic responses to balloon pumping. Be-
fore cardiac assistance, left ventricular end
diastolic pressure, pulmonary capillary
wedge pressure and pulmonary artery pres-
sure were severely elevated. Cardiac output
was normal. Lactate and pyruvate studies
indicated anaerobic cardiac metabolism.
With approximately 2 hr of cardiac assist-
ance, left ventricular pressure, pulmonary
capillary wedge pressure, and pulmonary
artery pressure returned to near normal.
Cardiac output increased by 16%. Lactate
extraction indicated aerobic myocardial
metabolism. Cardiac assistance was discon-
tinued for a period of 30 min. Left ven-
tricular end diastolic pressure and pulmo-
nary artery pressure rose significantly. After
resumption of diastolic augmentation for 3
min, hemodynamic parameters returned to
near normal values. Because of the favor-
able response to diastolic augmentation, the

Table 3.—Preoperative Hemodynamic Evaluation of Patient Using Temporary
Intraaortic Balloon Pump*

| Time | Balloon Pump Procedure | Pressure (mm Hg) | | | | Cardiac Output | Δ Eh § (mV) |
|------|----|-------|-------|-----------|-----|-----|--------|
| | | LV † | PCW | MPA ‡ | CVP | | |
| 18:10 | (Control) | 80/24 | (28) | 50/23 (33) | 10 | 5.5 | −2.92 |
| 18:15 | Balloon ON | | | | | | |
| 18:16 | Balloon ON | 80/24 | (25) | (31) | | | |
| 21:45 | Balloon ON | 86/10 | (14) | 24/10 (15) | 1 | 6.4 | + 2.22 |
| 22:25 | Balloon OFF | | | | | | |
| 23:00 | Balloon OFF | 90/16 | | 45/20 (27) | 4 | 4.9 | |
| 23:18 | Balloon ON | | | | | | |
| 23:20 | Balloon ON | 90/8 | (10) | 27/10 (18) | 1 | 5.3 | |

* Data collected on 8/4/71 following insertion of balloon pump (11:15 a.m.). It was removed at 8:05 a.m. on 8/5/71. Mechanical auxiliary ventricle was implanted 5 days later.

† Peak pressure/end diastolic pressure.

‡ Mean values given in parentheses.

§ $\Delta Eh = 30.7 \times \log \frac{\text{arterial lactate} \times \text{arterial pyruvate}}{\text{coronary vein lactate} \times \text{coronary vein pyruvate}}$ . Negative values of ΔEh indicate lactate production (anaerobic metabolism). Positive values indicate lactate extraction (aerobic metabolism).[12]

LV: left ventricle; PCW: pulmonary capillary wedge; MPA: main pulmonary artery; CVP: central venous pressure; CS: coronary sinus; ΔEh: myocardial oxidation-reduction potential.

patient was considered a good candidate for the mechanical auxiliary ventricle.

Five days later, on August 10, 1971, the mechanical auxiliary ventricle was implanted. The thoracic cavity was entered through the fourth intercostal space. When total cardiopulmonary bypass had been established, the descending thoracic aorta was cross-clamped proximally and distally, and a 15-cm incision was made into its lateral wall between the occlusive clamps; six pairs of intercostal arteries were temporarily occluded. The mechanical auxiliary ventricle was then sutured in place using a double row of continuous everting sutures reinforced with Dacron felt. A pacing electrode was sutured to the left atrium and a pacemaker was implanted subcutaneously in the left chest wall. The previously implanted transvenous pacemaker was disconnected. The pneumatic conduit was led out of the chest and positioned subcutaneously in the left hypogastric region where it could be connected via a permanent access button to the external drive unit. To provide a stable ECG signal for the external driving unit, electrodes were sutured to the apex of the left ventricle and the leads were brought out through the same access button. The dynamic aortic patch was activated and cardiopulmonary bypass was discontinued. The chest wall was closed in routine fashion.

Postoperatively, the patient's hemodynamic condition stabilized with a pulmonary artery pressure within normal ranges. Pulmonary capillary wedge pressure was elevated to 13–15 mm Hg. No evidence of right ventricular failure was present. During the first postoperative week, atrial fibrillation occurred intermittently, probably due to competition between the patient's own sinus rhythm and the implanted atrial pacemaker. Sinus rate was reduced by increasing doses of digitalis and a thiazide diuretic. The patient has since remained in atrial pacemaker rhythm with only occasional premature ventricular contractions. Except for this complication and a shallow dehiscence of the chest incision which was repaired under local anesthesia, the patient's postoperative course has been uneventful. Hematologic studies which included routine measurements of haptoglobin, urine hemosiderin, and microscopic red cell morphology showed no evidence of

Fig. 6.—Preoperative and day 21 postoperative chest X rays demonstrating reduction in cardiac transverse diameter.

hemolysis. Scanning electron microscopy with utilization of Nomarski optics and routine electron microscopy demonstrated no abnormal red cell or platelet morphology. Studies of platelet aggregation and blood coagulation were normal and consistent with the postoperative state. Heart size (transverse diameter) has decreased from 190 mm preoperatively to a normal 160 mm 21 days postoperatively (Fig. 6). The patient's exercise tolerance has increased, permitting him to walk several hundred yards without assistance. At the time of writing, 1 mo after the operation, it is planned to perform complete cardiac catheterization to document possible benefits of patch booster implantation. On recovering from this procedure, the patient is to be discharged.

## COMMENT

In the 4 yr since clinical trial of balloon pumping was initiated, evidence has been obtained suggesting that this technique can restore the acutely failing circulation to a stable operating point. The precise hemodynamic and physiologic effects of the procedure have not been completely defined, however, and assessment of the influence of balloon pumping on mortality in cardiogenic shock must await analysis of data on larger series of patients than have been accumulated to the present.

Balloon pumping is increasingly in use as a supportive modality for patients undergoing catheterization and cardiac surgery, particularly myocardial revascularization,[26,30,39] and a consensus may be developing regarding the effectiveness, low risk, and relative simplicity of the technique in this application.

A permanent mechanical auxiliary ventricle with a biocompatible blood interface appears to have been developed. This prosthesis, which was evolved from earlier configurations of the permanently implanted

in-series assist device, is intended to provide intermittent cardiac assistance during a period of months or years. Clinical data obtained in the initial case suggest that these effects may be sufficient to ameliorate medically intractable chronic congestive failure.

## SUMMARY

Laboratory and clinical experience with two techniques of in-series circulatory assistance is described. Intraaortic balloon pumping is a bedside procedure in which a catheter-mounted polyurethane pumping chamber having approximately the same volume as that of the failing left ventricle is passed through a femoral arteriotomy into the descending thoracic aorta. To obtain diastolic augmentation, the pumping chamber is inflated with helium at the beginning of ventricular diastole and deflated at the end of diastole.

In a clinical trial of this technique, our group has treated 40 patients in pharmacologically refractory cardiogenic shock secondary to acute myocardial infarction. Of 25 patients in whom shock began within 30 hr of the onset of symptoms of infarction, 23 (90%) came out of shock during balloon pumping. Circulatory stabilization was restored in 19 (75%), permitting mechanical assistance to be discontinued. Eleven patients (45%) recovered from their infarcts and were discharged from the hospital.

In the remaining 15 patients, the onset of shock was delayed beyond 30 hr after the acute infarction. Restoration of circulatory stabilization and discontinuation of balloon pumping were achieved in 12 of these patients, but none survived to hospital discharge. As yet, both the possible pathophysiologic basis for differentiating these two groups and the reasons for the uniformly fatal outcome in the second group remain undefined. It is possible that the latter group of patients is untreatable except by recourse to heart transplantation or permanent mechanical assistance.

The dynamic aortic patch is a permanently implanted mechanical auxiliary ventricle having the same operating principle, intraaortic location, and hemodynamic effects as the balloon pump. In its initial clinical trial, preliminary data suggesting its efficacy in alleviating severe, progressive chronic congestive failure were obtained. Continued clinical investigation utilizing both types of in-series assistance appears desirable.

## REFERENCES

1. Bregman, D., Kripke, D. C., Cohen, M. N., Laniado, S., and Goetz, R. H.: Circulation 41, 42: III-76, 1970.

2. Brown, B. G., Goldfarb, D., Topaz, S. R., and Gott, V. L.: J. Thorac. Cardiovasc. Surg. 53: 789, 1967.

3. Butner, A. N., Krakauer, J. S., Rosenbaum, A., Tjønneland, S., Sherman, J. L., Jr., Dresdale, D. T., and Kantrowitz, A.: Surg. Forum 20:199, 1969.

4. Clauss, R. H., Birtwell, W. C., Albertal, G., Lunzer, S., Taylor, W. J., Fosberg, A. M., and Harken, D. E.: J. Thorac. Cardiovasc. Surg. 41: 447, 1961.

5. —, Missier, P., Reed, G. E., and Tice, D.: In Dig. 15th Annual Conference on Engineering in Medicine and Biology, 4:44, 1962.

6. Corday, E., Swan, J. C., Lang, T. W., Goldman, A., Matloff, J. M., Gold, H., and Meerbaum, S.: Amer. J. Cardiol. 26:595, 1970.

7. Cronin, R. F. P., Moore, S., and Marpole, D. G.: Canad. Med. Ass. J. 93:57, 1965.

8. Feola, M., Norman, N. A., Haiderer, O., and Kennedy, J. H.: In Hastings, F. W., and Harmison, L. T. (Eds.): Artificial Heart Conference Proceedings. Washington, D. C., Artificial Heart Program, 1969, p. 637.

9. Friedberg, C. K.: In Diseases of the Heart (ed. 3). Philadelphia, Saunders, 1966, p. 801.

10. Gallo, E., Eichelter, P., and Schenk, W. G.: J. Thorac. Cardiovasc. Surg. 52:745, 1966.

11. Goldfarb, D., Brown, B. G., Conti, C. R., and Gott, V. L.: J. Thorac. Cardiovasc. Surg. 55: 243, 1968.

12. Gudbjarnason, S., Hayden, R. O., Wendt, V. E., Stock, T. B., and Bing, R. J.: Circulation 27:937, 1962.

13. Jaron, D., Freed, P. S., Phillips, S., Zorzi, G., and Kantrowitz, A. (in press). *In* Proceedings of 24th Annual Conference on Engineering in Medicine and Biology Soc. Eng. Med. Biol.

14. —, Tomecek, J., Freed, P. S., Welkowitz, W., Fich, S., and Kantrowitz, A.: Trans. Amer. Soc. Artif. Intern. Organs 16:466, 1970.

15. —, et al.: *In* Proceedings of 21st Annual Conference on Engineering in Medicine and Biology 10:20.2, 1968.

16. Kantrowitz, A., Akutsu, T., Chaptal, P. A., Krakauer, J., Kantrowitz, A., and Jones, R. T.: JAMA 197:525, 1966.

17. —, and Kantrowitz, A.: Surgery 34:678, 1953.

18. — et al.: Progr. Cardiovasc. Dis. 12:293, 1969.

19. —, Phillips, S. J., Butner, A. N., Tjønneland, S., and Haller, J. D.: J. Thorac. Cardiovasc. Surg. 56:219, 1968.

20. —, Krakauer, J., and Sherman, J. L., Jr.: J. Cardiovasc. Surg. 9:1, 1968.

21. —, Tjønneland, S., Freed, P. S., Phillips, S. J., Butner, A. N., and Sherman, J. L., Jr.: JAMA 203:113, 1968.

22. —, et al.: Trans. Amer. Soc. Artif. Intern. Organs 14:344, 1968.

23. Krakauer, J. S., Rosenbaum, A., Freed, P. S., Jaron, D., and Kantrowitz, A.: Amer. J. Cardiol. 27:123, 1971.

24. Laird, J. D., Jones, R. T., Austen, W. G., and Buckley, M. J.: *In* Proceedings of 20th Annual Conference on Engineering in Medicine and Biology 9:26.6, 1967.

25. Lee, H. L., Jr., Ocumpaugh, D. E., Cupples, A. L., Culp, G. W.: Report No. PH43-67-1108-1. Artificial Heart Program. U.S.P.H.S. National Institutes of Health, Bethesda, Md., 1970.

26. Leinbach, R. C., Buckley, M. J., Austen, W. G., Petschek, H. E., Kantrowitz, A. R., and Sanders, C. A.: Circulation 41,42:III-76, 1970.

27. Moulopoulos, S. D., Topaz, S., and Kolff, W. J.: Amer. Heart J. 63:669, 1962.

28. Mueller, H., Ayres, S., Conklin, F., Giannelli, S., Jr., Mazzara, J., Grace, W., and Nealon, T.: J. Clin. Invest. 50:1885, 1971.

29. —, —, Mazzara, J., Gianelli, S., Jr., Conklin, F., and Grace, W.: Circulation 41,42:III-81, 1970.

30. Mundth, E. D., et al.: Surgery 70:78, 1971.

31. Nachlas, M. M., and Siedband, M. P.: J. Thorac. Cardiovasc. Surg. 53:698, 1967.

32. Powell, W. J., Jr.: Amer. J. Cardiol. 25:122, 1970.

33. Scheidt, S., Ascheim, R., and Killip, T.: Am. J. Cardiol. 26:556, 1970.

34. Schilt, W., Freed, P. S., Khalil, G., and Kantrowitz, A.: Trans. Amer. Soc. Artif. Intern. Organs 13:322, 1967.

35. Sharp, W. V., Gardner, D. L., and Andresen, G. J.: Trans. Amer. Soc. Artif. Intern. Organs 12:179, 1966.

36. —, McVay, W. P., Flaksman, R. J., and Wright, J. I.: Surg. Forum 20:141, 1969.

37. Soroff, H. S., Giron, F., and Ruiz, J.: New Eng. J. Med. 280:693, 1969.

38. Sujansky, E., Tjønneland, S., Freed, P. S., and Kantrowitz, A.: Surgery 66:875, 1969.

39. Summers, D. N., et al.: Arch. Surg. (Chicago) 99:733, 1969.

40. Talpins, N. L., Kripke, D. C., and Goetz, R. H.: Arch. Surg. (Chicago) 97:991, 1968.

41. Tanaka, S., Rassman, W., Lande, A., Kaster, R., and Lillehei, C. W.: J. Thorac. Cardiovasc. Surg. 58:112, 1969.

42. Tomecek, J., Jaron, D., Tjønneland, S., and Kantrowitz, A.: Trans. Amer. Soc. Artif. Intern. Organs 15:406, 1969.

43. Tyberg, J. V., Keon, W. J., Sonnenblick, E. H., and Urschel, C. W.: Amer. Heart J. 80:89, 1970.

44. Urschel, C. W., Eber, L., Forrester, J., Matloff, J., Carpenter, R., and Sonnenblick, E.: Amer. J. Cardiol. 75:546, 1970.

45. Yahr, W. Z., Butner, A. N., Krakauer, J. S., Tomecek, J., Tjønneland, S., and Kantrowitz, A.: Surg. Forum 19:142, 1968.